

# **STUDY OF OCULAR CHANGES IN DIABETES MELLITUS IN BUNDEL -KHAND REGION**

## **THESIS FOR MASTER OF SURGERY [OPHTHALMOLOGY]**



**BUNDELKHAND UNIVERSITY  
JHANSI (U.P.)**

*Approved  
D. 11/11/97  
3/1/98*

1997

ARJUN SINGH SARANG

DEDICATED

TO

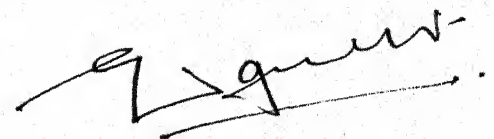
HUMANITY

## **CERTIFICATE**

This is to certify that the work entitled "*STUDY OF OCULAR CHANGES IN DIABETES MELLITUS IN BUNDELKHAND REGION*", which is being submitted as a thesis for M.S. (Ophthalmology) Examination, 1997 Bundelkhand University, Jhansi by Arjun Singh Sarang, has been carried out in the Department Ophthalmology, M.L.B. Medical College, Jhansi.

He has put in necessary stay in the department as required by the regulations of Bundelkhand University, Jhansi.

Date : 17-1-97



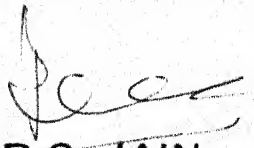
**Dr. G.D. GUPTA**  
M.S., D.O.M.S.

*Associate Professor & Head,  
Department of Ophthalmology  
M.L.B. Medical College,  
JHANSI, (U.P.)*

## **CERTIFICATE**

This is to certify that the work entitled "*STUDY OF OCULAR CHANGES IN DIABETES MELLITUS IN BUNDELKHAND REGION*", which is being submitted as a thesis for M.S. (Ophthalmology) Examination, 1997 Bundelkhand University, Jhansi, has been carried out by Arjun Singh Sarang, under my guidance and supervision. The techniques and observations incorporated in this thesis have been undertaken by candidate himself and checked by me from time to time.

Date : 17-1-97

  
**Dr. B.S. JAIN**  
M.S.

Associate Professor,  
Department of Ophthalmology  
M.L.B. Medical College,  
JHANSI, (U. P.)

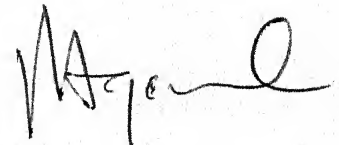
**(GUIDE)**



## **CERTIFICATE**

This is to certify that the work entitled "*STUDY OF OCULAR CHANGES IN DIABETES MELLITUS IN BUNDELKHAND REGION*", which is being submitted as a thesis for M.S. (Ophthalmology) Examination, 1997 Bundelkhand University, Jhansi, has been carried out by Arjun Singh Sarang, under my guidance and supervision. The techniques and observations incorporated in this thesis have been undertaken by candidate himself and checked by me from time to time.

Date : 17-1-97



**Dr. Navneet Agarwal**  
M.S.

*Assistant Professor ,  
Department of Medicine  
M.L.B. Medical College,  
JHANSI, (U. P.)*

**(CO-GUIDE)**

### ACKNOWLEDGEMENT

I wish to express my gratitude to all of them who helped me to complete the present work.

I am indebted to my teacher and guide Dr. B.S. Jain, M.S., Associate Professor, Department of Ophthalmology, M.L.B. Medical College, Jhansi for giving me the privilege to work under his affectionate guidance. The present work at every stage bears the impressions of his valuable suggestions, constructive criticism and meticulous attention. I am extremely grateful for the ceaseless attention he had paid to my problems, and constant encouragement throughout this work. He inspired me with his precisions in work, untiring zest and unfathomed knowledge throughout his humble venture.

I fail to express my sense of indebtedness from the deepest recess of my heart to my esteemed benevolent teacher Dr. G.D. Gupta, MS, DOMS, Associate Professor and Head of the department of Ophthalmology, M.L.B. Medical College, Jhansi for helping me the way out to my problems during the course of my study and for teaching me the fundamentals so that I could continue to learn for the rest of my life.

I would also like to thank and express my obligations to my co-guide Dr. Navneet Agarwal, MD, Assistant Professor, Department of Medicine, M.L.B. Medical College,

Jhansi for his extreme concern and kindness towards me and my work, who has generosity allowed me to see patients under his care.

I would like to acknowledge my indebtedness to Dr. V.K. Misuria, MS, Assistant Professor, Department of Ophthalmology, M.L.B. Medical College, Jhansi who have helped me the way out to my problems during the course of my study work. I am undoubtedly thankful to my senior resident Dr. Chitra Singh who has helped me on every step in building up the infrastructure of this work. I am thankful to my colleagues and my friends especially Dr. Anurag Garg, Dr. Kapil Paliwal for boosting my confidence.

Finally, I must say a necessary work of appreciation for those who took immense care and patience in typing out this manuscript.

Dated : 17-1-97

  
( ARJUN SINGH SARANG )

# C O N T E N T S

CHAPTER		PAGE NUMBER	
		From	To
INTRODUCTION	...	1	3
REVIEW OF LITERATURE	...	4	27
MATERIAL & METHODS	...	28	32
OBSERVATION	...	33	52
DISCUSSION	...	53	66
CONCLUSION	...	67	70
BIBLIOGRAPHY	...	71	81
SUMMARY	...	82	attached <sup>83</sup> )

# INTRODUCTION

## INTRODUCTION

Diabetes mellitus is the most common endocrine disease. The true frequency is difficult to ascertain because of differing standards of diagnosis but probably it is between 1 and 2 percent. The disease is characterized by metabolic abnormalities; by long-term complications involving the eyes, kidneys, nerves and blood vessels; and by a lesion of the basement membranes demonstrable by electron microscopy.

The first clinical description and the name of diabetes was given almost two thousand years ago by Aretaeus of Cappadocia.

There has been a greater increase in the number of diabetics in the past few decades than can be explained by the general increase in average age of the population. In the United States, diabetes statistically holds seventh place as a cause of death and third place as a cause of blindness.

Insulin dependent (Type I) diabetes is due to damage to the Beta cells of Pancreatic islet of Langerhans. It is not directly inherited, although individuals may inherit a predisposition associated with certain HLA types. The peak incidence is 10 - 20 years, although elderly patients can also be Insulin dependent. Non-insulin dependent



(Type II) diabetes has no known cause, although in many causes there is a strong genetic component, unrelated to the HLA system. It is most prevalent after middle age and occurs most frequently between the ages of 50 and 70 years, although there is a certain amount of overlap between the two types of diabetes.

The prognosis of the diabetic patient has basically changed with the discovery of insulin. Diabetic coma was the main cause of death in diabetics before 1921, and the average time of survival was about 5 years. Today, a diabetic lives almost as long as the average normal person. His Fate is extensively determined by the late complications of the blood vessels. Involvement of small as well as large blood vessels presently represents almost 80% of the direct cause of death in the diabetic patient. They should, therefore, be the main object of the medical and scientific effort.

Diabetic microangiopathy is virtually specific for diabetes mellitus according to Jaeger (1856). Diabetic retinopathy represents on direct result of this angiopathy. Arteriosclerosis and hypertension may predispose for the development of diabetic retinopathy. Diabetes on the other hand, may cause earlier development of arteriosclerosis and hypertension.

Vision is significantly affected due to functional or structural damage of different structures of the eye. These changes are related to duration of disease, age of onset and control of diabetes.

The diabetes in eyes creates problems in two different ways, one by affecting the body functions as a risk factor, secondly by changing the osmolarity of blood and tissue hydration which leads to rapid and repeated changes in number of glasses, blurring of vision, muscle weakness, recurrent infection of lid, changes in lens and retina alongwith raised intraocular pressure. Among these diabetic retinopathy is a serious complication which leads to blindness.

REVIEW OF LITERATURE

## REVIEW OF LITERATURE

Complications of Diabetes mellitus are known to occur in almost every part of the visual organ. Some of these changes are of no importance and are not characteristic. Other, however, are pathognomonic for diabetes. Statistical data about the incidence of blindness due to its ophthalmic complications is 20 times more than the incidence of blindness in general population. According to data from statistics on blindness in model reporting area, 1962-70, diabetic retinopathy was responsible for 11.1% of the new cases of legal blindness in all the age groups and 19.1% of those in the 20 to 64 year age group.

The ocular changes related to diabetics are :

### Lids :

Blepharitis, recurrent Hordeolum and eczema with poor healing tendencies should always place diabetes as a cause on the list of differential diagnostic possibilities. In diabetics, simple infections may take a serious course. Gangrene of the lid following hordeolum or orbital cellulitis in dacryocystitis are examples.

### Conjunctiva :

Aneurysms of the conjunctival blood vessels are found in 55% of diabetics (Mc Culloch and Pashby 1950). The significance is disputed. We agree with Velhagen 1943,

who stated that these conjunctival changes allow no conclusions as to type and stage of the diabetes.

Cornea :

This is affected in several ways, A classic sign is presence of corneal striae or folds of Descemet's membrane sometimes called Beetham's lines, secondary to severe ocular hypotony Waite and Beetham 1935. However, these changes are not specific for diabetes. Staining of the cornea was found by Janert to be more common in diabetics. Epithelial keratodystrophy was published in Italian literature by Quaranta 1954 and confirmed by Iolispada in 1864. The decreased corneal sensitivity was first demonstrated by Schullica and Proto and confirmed Schwartz in 1974, decrease in corneal sensitivity is believed to be part of a generalized polyneuropathy that develops in diabetes mellitus (Schwartz 1974). The neuropathy mechanism is known but on ischemic angiopathy of vasa nervorum or other small vessels may be involved (Woltman, Wilder 1929).

A demyelinating process may exist, perhaps related to chronic schwann cell dysfunction or alteration in the level of nerve free myoinositol content may also contribute to the neuropathy (Thomas 1966).



Awasthi et al (1974) have recently shown that the reduction in corneal sensitivity is more significant in patients with proliferative retinopathy. Recurrent corneal erosion which do not heal rapidly is seen (Joslin 1978). Deficient tear production and basement membrane abnormalities may also contribute to epithelial breakdown or epithelial healing problem in some diabetic patients (Mandelcorn 1976). In a study of 100 cases by Prakash Kannan and Ananda noted following corneal changes like decrease in corneal sensitivity. Epithelial defects, erosions, neurotrophic ulcers, stroma increase in corneal thickness, opacities, Descemet's membrane wrinkles, Endothelium undergoing polymegathism, pleomorphism, pigment deposits. Deep corneal opacities were also observed by Noothoven Van Goor and Schaly (1943).

#### Iris :

Diabetes uniformly affects the iris. The presence of diabetes in life can be inferred by the finding of glycogen deposition in pigment epithelium of the iris in post-mortem specimens, (Becker 1883, Waite 1935). Diabetic iritis was first described by Marchal in 1863 and occurs in 0.8 - 8% of cases. Elschmig 1937 differentiates a metastatic type of iritis with a severe course from a different type that is caused by destruction of pigment and



tend to cause glaucoma. Hydropic degeneration of the pigment epithelium is the cause of the black staining of the aqueous humour during intraocular surgery (Armaly and Baloglov 1967).

Diabetic rubeosis iridis was first described in 1876 by Abadie and was named by R. Salus 1928. This is characterized by neovascularization in the pupillary portion of iris and in the chamber angle. Rubeosis develops in iris without signs of inflammation. This was also explained by Kurz 1932 and Francois 1951. Fibrovascular membrane develop causing radial contraction with the result that the pupil is distorted and the posterior iris pigment epithelium pulled through the pupil into the anterior surface of the iris. Ectopic uveae, this membrane may grow further producing pupillary membrane (Schulze 1967). As these new vessels external into the angle they, along with fibrous membrane, which accompanies them, start to occlude the aqueous outflow channel leading to increase intraocular tension (Smith 1954). Occasionally the new vessels disappear spontaneously and with them the raised tension. Fixed pupils may occur on one or both sides in diabetics. Glycogen infiltration into the epithelial cells and subsequent degeneration in iris epithelium may be responsible for diabetic pupil to respond delay to myetriatics (Duke Elder 1940).

Lens :

The first report of diabetics relation with cataract was given by John Rallo (1798). While by the time Bendt (1834), T Bendict (1842) and Himly (1843) have also reported that the patients who are diabetic they are prone to develop cataract.

The percentage of patients representing for surgical treatment of cataract has been found to be higher among diabetic than non-diabetics (Clegg 1920), Anthonisen 1936, Towners and Carey 1955, Norm 1967). All found that about 5% of patients who have had cataract extraction were diabetics. Caird et al 1964 and Pirie 1965 found 3% of all patients who came to cataract OPD were having diabetes.

Kirby 1933, found that 64% of all the diabetics has lenticular changes of some kind of which 70% were of senile cataract type. 21% nuclear, 7% posterior cortical and 2% subcapsular type. Lenticular opacities may appear at a relatively early age in people in the prediabetic state (Pautique and Michand 1965; Buckner 1965; Van Salm 1966). Caird, Pirie and Ramsell 1969 noted 4 to 6 times cataract more likely to develop at a young age and to progress more rapidly. The recorded proportion of juvenile diabetics having opacities varies from 2 to 5% (Keim 1950, Janert 1956, Mohriake 1956, Gunther 1956, Porsium et al 1964, Knowles

et al 1965, Burdi Et and Caird 1965) found a frequency of 5% and 11% after 10 years of diabetes and 23 to 32% after 20 years.

Classically the diabetic or floccular cataract consists of showers of small granular opacities (Christmas tree pattern). These appear as "snow flake" dots directly under both the anterior and posterior lens capsules. Van Heyningen (1950) sought the pathogenesis of metabolic cataract he said increased concentration of glucose enter the lens and activate the enzyme aldose reductase which favours the conversion of glucose to sorbitol. Sorbitol is not promptly metabolized but through osmotic effect draws water into the lens bringing with it sodium, which damages the lens fibers and result cataract formation.

The risk of cataract associated with diabetes is greater in females than in males (John J harding 1993). It was seen that cataract extraction is relatively commoner in diabetic women than in men specially under the age of 70 years according to study by Pules 1956, Fitzgenald et al 1961.

Perkins ES (1983) incidence of diabetes in cataract series was 13.66%. The higher ratio of women to men over in the diabetes was particularly striking and the age at which the surgery was performed was significantly lower in both men and women than in non-diabetics.

Statistics of blindness registration for cataract thought to be diabetic in origin are few in number and are subject to all the reservations in relation to blindness registration for cataract as a whole. Data from Canada 1865 and from a small study in west of Scotland (Committee on blindness 1910) suggest that blindness from cataract in diabetics total about 25 - 33% of that due to diabetic retinopathy and represents about 2% of all blind registrations of diabetics registered blind from cataract 70% are over 70 years old and 81% are women.

Cataract extraction in the diabetic carries increased risk of rubeosis iridis, neovascular glaucoma, acceleration of proliferative diabetic retinopathy with or without vitereous haemorrhage and difficulty in corneal wound and epithelial healing.

There is good agreement that the visual results of cataract extraction are almost good in diabetics without retinopathy as in non-diabetics. About 80% of both groups will gain vision of 6/12 or better. Where if retinopathy is present only 34% of patients will achieve this acuity. Retinopathy is main reason for poor visual results after cataract extraction in diabetics, as other ocular diseases are in non-diabetics (Caird et al 1965). Retinopathy is by no means an absolute contraindication to operation even



in presence of quite severe retinopathy and when the visual acuity is not greatly improved the patient may obtain visual benefit from operation.

Diabetic patients have the same range of refractive errors as non-diabetics but there is some evidence that they become presbyopic at a younger age than person in general population. Quite often the presenting sign of diabetes is myopia induced by hyperglycemia. Diabetic patients often state that vision is less clear at certain times of the day because refractive errors change alongwith blood glucose. So eye glasses should not be prescribed during an acute illness. Diabetics often complain of visual disturbances in association with hypoglycemic episodes. These are not refractive errors but are commonly due to CNS effects of hypoglycemia.

#### Colour vision :

A possible association between loss of colour vision in patients with diabetes mellitus was first reported in 1905 (Roy et al 1926). The first controlled study of colour vision in diabetic patients was reported in 1972 and 1973 by Knnear and Lahowski and Colleagues who showed in large group of scottish diabetic patients that blue, yellow and blue green colour vision losses were found significantly more among the diabetic patients with retinopathy than in normal controls.

Other more recent studies, using controls (Lombrail et al 1984) and normative data from Belgium (Condit R and Breenik et al 1982-1984) have confirmed that colour vision is significantly altered in diabetic patients with advanced diabetic retinopathy.

However, it still remains unclear whether or not colour vision losses are already present in diabetic patients who have little or no diabetic retinopathy.

Dubois poulson and Cochet and Verriest 1954 to 1964 were first to note, in case reports, and alterations of colour vision in diabetic patients without retinopathy. Thus clinical data shows no significant association between presence or absence of a colour vision defect and age, sex age at onset or duration of diabetes. Some studies reveal insulin dependent diabetics patients with no to minimal diabetic retinopathy had significantly more colour defects than controls. In all these studies it is important to exclude diabetic patients with any type of lens changes.

#### Intraocular pressure in Diabetes :

It was Heine (1903) and Krause (1904), who first observed a striking hypotony of the eyeball during diabetic coma. Crafe (1924) and Poos (1930) drew attention to the extreme variation in blood sugar levels, considering that these reacted on the intraocular pressure when conditions



for a glaucoma were present. Igersheimer (1944) Philips (1946), Crtiz (1947, 1948), Weinstein (1948) and Larsen (1960) have all stressed how a changing intraocular pressure could as a mechanical factor, initiate or favour the development of diabetic retinopathy.

Armastrong, Dailly, Dobsen and Girord (1960) on the basis of a relatively permanent intraocular pressure of 23.4 mmHg or higher (Schiotz Tonometer) have recently found an incidence of glaucoma in diabetes of atleast 6.6% while previously Waite and Beetham in 1935 and reported only 0.5% of clinical glaucoma in 2002 diabetic patients.

The mean intraocular pressure in diabetics is 19.25 mmHg (Arora and Prasad 1983) which is higher than the normal mean intraocular pressure reported in general population i.e. 16.1 mmHg (Becker and Schaffer). While in Juvenile diabetics the mean intraocular pressure through lower 17.93 mmHg than the mean intraocular pressure in maturity onset diabetes mellitus, was higher than the average normal mean intraocular pressure. However, Palmar (1935) and Armaly and Bologlous (1967) observed low intraocular pressure in diabetics than non-diabetics.

Intraocular pressure in reaction to different grades of retinopathies. Christiansons (1960) studied 172 diabetic

presence between 12 - 50 years of age, the aim was to observe the reaction of diabetic disease on the pressure of eye, while ignoring as far as possible the influence of age. He found that the diabetic retinopathy was set up only in 45.6% cases, a similar co-relation was found by Kornurup (1955) i.e. 48.6%. He also reported that the diabetic eye have a higher intraocular pressure than a corresponding normal eye. Insulin treatment seems to have but little relevance as regards this difference in tension, with increasing retinopathy, difference in tension is accentuated.

Christianson found that in grade I retinopathy IOP was 16.1 mmHg on an average mean, in grade II, the tension decreased to 12.3 mmHg and in grade III still decreased to 9.3 mmHg. He also reported eye with diabetic retinopathy of grade IV have lower intraocular pressure than other retinopathic group and the co-efficient of facility of outflow rises.

Igershemier (1944) suggested that ocular hypotony in diabetes might play a role in development of diabetic retinopathy, it has also been demonstrated that diabetic retinopathy often progress rapidly in such ocular hypotonic states as pregnancy and after eye operations (Lieb WA 1967, Radan et al 1968).

A number of studies have pointed out a greater occurrence of proliferative retinopathy has been appreciated in diabetics with consistently low intraocular pressure than those with elevated intraocular pressure (Igersheimer 1944) or conversely a greater occurrence of proliferative retinopathy is extremely rare in diabetic individual with primary open angle glaucoma.

Topical corticosteroids have been demonstrated to induce increase intraocular pressure in human eyes. The degree of pressure response was genetically transmitted in Mendelian fashion of particular interest was close relationship between this genetically determined response and primary open angle glaucoma.

Becker and Khan (1964), suggestions have been made that elevated intraocular pressure might be used to prevent the patients with diabetes from developing proliferative retinopathy.

#### Diabetic Retinopathy :

Diabetic retinopathy first described by Von Jaeger in 1855 is one of the major tragedies of ophthalmology, in our present generation it is predictable but not preventable. Chronic and progressive in its course and leading to blindness in interesting percentage of cases. Desmarres (1856) AV Graefe (1859), T Leber (1875), S McKenzie and E. Nettleship (1877) J. Hirschberg (1890) and many others described the clinical picture of diabetic retinopathy in gross outlines.

In the insulin era after 1923, the relation between the clinical picture and the morphologic findings in diabetic retinopathy were studied by Ballantyne and Lowenstein (1943), Friedenwald (1943), Ashton (1949), Thiel (1956), Cogan, Kuwabara, Toussaint (1961), Wolter, Bloodworth (1961) Patz and Maumence (1962).

Diabetic retinopathy has been divided into three stages according to fundus changes seen after dilation of pupils (Lloyd, Aiello et al 1978).

Stage I :

Background retinopathy which includes presence of microaneurysms with or without small dot & blotch haemorrhages, hard exudates and fewer than five soft exudates, minor venous abnormalities characterized by irregularities in the width of veins, sheathing of veins, tortuosity of veins, arteriolar narrowing, arteriovenous nicking, retinal oedema.

Stage II :

Preproliferative diabetic retinopathy which includes presence of cotton wool spots more than five, venous beading and duplication, intra retinal microvascular abnormalities. Areas of nonperfusion or capillary closure, macular odema in young patient.



Stage III :

Proliferative diabetic retinopathy which includes -

- i) Vasoproliferation new vessels on the disc or elsewhere fibrous tissue membrane.
- ii) Fibrous growth stage with contraction leading to retinal haemorrhage, vitreous haemorrhage, tractional retinal detachment.

Mekenzic and Nettleship (1877) were the first to discover capillary aneurysms in a case of Glycosuria, Ballantyne and Loewenstein (1943) succeeded in proving that the earliest sign in diabetic retinopathy is the microaneurysm. These seen ophthalmoscopically are small size round in shape, sharply defined and sometime show a light reflex which iridicate spherical form, these may be confused with punctate haemorrhage which are not globular but petechial in shape. The microaneurysms can be found in any part of fundus but when few usually near the macula. They often seem to be attached to five perimacular vessels with line. These undergo sclerosis or complete hyalinisation with or without thrombosis leading to formation of opaque scar which appears as surrounded by a fank narrow hale or white spot.

Haemorrhages, most characteristically occuring in the deeper layers of ratina and hence round and regular in

shape and are also a relatively early feature of diabetic retinopathy (Ballantyne and Lowenstein 1943). These may occur due to rupture of microaneurysms which are at level of ganglion cell layer and punctate shape. These are grouped under dots and blots.

The exudates usually situated in intranuclear layer but later breaking down barriers between these space and forming compact masses waxy patches. These differ from that of hypertensive exudates which is fibrinous, large patch granular appearance and of a silvery grey colour while of diabetics the exudates are of fatty or lipid substance, smooth homogenous texture of hyaline or fatty deposits.

Hard exudates are more common. As regards the retinal vessels their contest clinical change is probably a general fullness of the larger veins (Ballantyne and Cowanster 1943, Michaelson 1949).

Phlebosclerosis formation of loops and cells are new built preretinal vessels O' Brien and Allen 1940, Philips 1946 are pathogenomic of diabetics. Network of capillaries (Retemirabile) which occur in all conditions characterized by circulatory statis in the retina are common in diabetics.



The pre retinal vascular proliferation may remain there or regress leaving a vitreo-retinal scar, frequently, however it invades the vitreous space either through gaps in the cortical vitreous (Tolentino et al 1966) or else, because it is pulled forward by a shrinking vitreous (Davis 1966). The proliferative phase of diabetic retinopathy is a true neovascularisation with the accompaniment of connective tissue (Dobree 1968). These new vitreal vessels arising from the intraretinal vessels do not have intramural pericyte cells (Hogan 1967). The contraction of the connective tissue may give rise to retinal detachment (Dobree 1968). Proliferative retinopathy is found in about 2 to 16 percent of diabetics (Wilson et al 1957; Postmann 1954; Engelson 1954; Scott 1951-53; Kornerup 1958).

Fluorescein photography helps to understand the pathological process, by which we can demonstrate capillary dilatation disturbance of normal capillary pattern which are among the earliest sign of diabetic retinopathy (Kohner 1967).

The development of disturbance of microaneurysm may take place in less than a year (Keen and Smith 1959). Exudates and haemorrhage may also come and go.

Incidence of Diabetic retinopathy :

The general increase in diabetes mellitus is high for its affects between 1-4 to 1.7% of population. It occurs particularly in people in fifth and sixth decade of life. There has be continous increase in the incidence of diabetic retinopathy in past few decades.

Wagenar figure of this point are; in 1921 immediately prior to the introduction of insulin, found an incidence of 8.5% of diabetic retinopathy among diabetics (Wagener and Wilder 1921). In 1934, the incidence has risen to 17.7% (Wagener et al 1944) and in 1945 - 29.6% (Wagener 1945). Typical figures based on large number of patients are those of Kornuruf (1957) who found 601 cases of diabetic retinopathy in 1285 unselected diabetics (47%) and Dollfus (1945) 681 cases in 1,303 patients (52.4%). It follows that, at present time diabetic retinopathy may be expected to develop atleast in 50% of all the cases of diabetes. In evaluating the statistics one has to consider the facts that until the introduction of insulin in 1921 many patients died before the occurrence of retinopathy, while thereafter, they continued to live to develop retinopathy and also that examining techniques are much more precise today than they were years ago. This is due to the improvement of ophthalmoscope and slit lamp and fluorescein angiography.

Diabetic retinopathy in relation to the age :

The first report of diabetic retinopathy by V Jaegas 1954 was based on the finding in a 22 year old, gardener. However, most workers have stated that retinopathy is common in patients of middle age group or advance age and rare in younger people and extremely rare below the age of 10 years whatever the duration of diabetes may be (Forsyth and Payne, 1956, Imerslund 1959, Girner 1960, Guest Lample Kessler and Skillman 1965).

Diabetic retinopathy in relation to duration of diabetes :

The duration of diabetes is the most common single factor for the causation of diabetic retinopathy. There is general agreement that the prevalence of retinopathy in the diabetic population is positively associated with the duration of diabetes. In the recent study by Kahn HA and Brodley RF 1975), the prevalence of diabetic retinopathy among patients at Joslin clinic, was 25% in total diabetic population, 7% in patients with diabetes for less than 10 years, 26% in patients with diabetes for 10 to 14 years, and 63% in patients with diabetes for 15 years or more. Retinal changes are rarely seen until the diabetic has been in existence for 3 years, a fact confirmed by all observers (Waite and Beetham 1935; Wagener 1945; Friedenwald 1950; Lawrance et al 1951; Gardes 1953; Scott 1953 and many Others). Patients who develop diabetes before the age of 15 or 16 show a frequency of 10% or less after 5-9 years of diabetes, of



Diabetic retinopathy in relation to the age :

The first report of diabetic retinopathy by V Jaegas 1954 was based on the finding in a 22 year old, gardener. However, most workers have stated that retinopathy is common in patients of middle age group or advance age and rare in younger people and extremely rare below the age of 10 years whatever the duration of diabetes may be (Forsyth and Payne, 1956, Imerslund 1959, Girner 1960, Guest Lample Kessler and Skillman 1965).

Diabetic retinopathy in relation to duration of diabetes :

The duration of diabetes is the most common single factor for the causation of diabetic retinopathy. There is general agreement that the prevalence of retinopathy in the diabetic population is positively associated with the duration of diabetes. In the recent study by Kahn HA and Brodley RF 1975), the prevalence of diabetic retinopathy among patients at Joslin clinic, was 25% in total diabetic population, 7% in patients with diabetes for less than 10 years, 26% in patients with diabetes for 10 to 14 years, and 63% in patients with diabetes for 15 years or more. Retinal changes are rarely seen until the diabetic has been in existence for 3 years, a fact confirmed by all observers (Waite and Beetham 1935; Wagener 1945; Friedenwald 1950; Lawrance et al 1951; Gardes 1953; Scott 1953 and many Others). Patients who develop diabetes before the age of 15 or 16 show a frequency of 10% or less after 5-9 years of diabetes, of

50% after 15 years or so and 80-90% after 26 years or more. Lundback (1955) on the initial examination of 246 recently diabetics, in whom the disease had presumably developed above the age of 40 years, found retinopathy present in only 4% whereas Dollfus and Haige (1953) found that 90% of diabetics of over 18 years standing had retinopathy and Dolger 1947 examining cases of 25 years standing, concluded that not a single case had escaped.

Cristiansson's (1961) reported 45.6% of the patients had some form of retinopathy in an average diabetic duration of 16.7 years, a frequency that is in close agreement with the figure given by Kornurup (1955), Hamely 46.8% in large patients. On the other hand patients with proliferative retinopathy Grade III and IV (Ballantyne 1946) in Christianssons study shows 17.5% of the total case material with an average diabetic duration of 16 years the corresponding figure of Kornurup (1958) are 8.4% with the duration of 16.1 years the higher frequency in Cristianssons study is due to age limit being fixed to 50.

But it is of interest to note that not all the diabetics with long duration of diabetes exhibit the retinopathy. Kinsell (1955) reports that Jeslin was able to reward 45 patients with "Quarter century victory model" in that they exhibited no signs of "Late diabetes syndrome"

even after 25 years of duration of diabetes. According to Friedenwald (1954), there are about 15-20% of diabetics who do not show diabetic retinopathy after 20-25 years of diabetes.

Khosla et al (1984) found that more severe form of retinopathy was seen as the duration of diabetes increased ( especially after 10 year period ) but contrary to the usual, the prevalence of diabetic retinopathy is related to the duration of diabetes.

Sex incidence of Diabetic retinopathy :

Braun (1937), Hanum (1988), Heinsiun (1939) and many Others concluded that diabetic retinopathy is more common in females than males. Duke Elder describes that females - male ratio is 3 : 2, and also that women are more liable to develop retinopathy. Proportion of female-male ratio with diabetic retinopathy is about 4 : 3; while in some other studies there is much greater incidence among females; Hanum (1938), for e.g. in 183 cases of diabetic retinopathy found 72% females and 28% males. The larger statistics of Postsmann and Wiese (1954), Keiding et al (1952). Janort et al (1956) and Babel and Rilliet (1958), however did not show a difference between the sexes.



### Frequency of Retinopathy and severity of the disease :

Retinal lesions are observed in mild as well as severe cases. Hanum (1938) found retinopathy to be most common in diabetics of mild to moderate severity. Scott (1957) also believes that retinopathy is more common in light diabetes. Donoghass and Drury (1954) in contrast to this found retinopathy to be more common when more insulin was required to control the diabetes. Mohmike also shows that retinopathy occurs earlier and more commonly in severe cases. Waite and Beetham (1935) however, finds no relationship at all between the frequency of retinopathy and the severity of disease.

### Effects of control of diabetes on retinopathy :

The clinical and experimental evidence suggests that good control of metabolic aspects of diabetes delays the onset and decreases the severity of retinopathy to prove this prospective study of metabolic control has been performed in experiment with animals (Engerman, Bloodworth 1973). They found that poorly controlled group developed retinopathy while striking reduction in incidence and severity of retinopathy in well controlled group.

### Diabetic retinopathy in relation to type of diabetes :

The familiar text book classification of diabetes into 'Juvenile onset' and 'maturity onset' has now been



largely abandoned by diabetologists, the classification adopted by the Americans Diabetic Association and W.H.O. divides the majority of patients into type I (Insulin dependent diabetes mellitus IDDM) and type II (Non-insulin dependent diabetes mellitus (NIDDM)). Abundant evidence show that these two have entirely different etiopathogenesis. Majority of type I patients develop the disease in childhood or in adolescence, but it is by no means confined to this age group. Type II predominantly affects the adults life. Etiology of type I is immune mediated destruction of Islet B cells, while etiology of type II remains mainly unknown.

In Juvenile diabetes, retinopathy rarely occurs before 16-18 years of age (Larson 1960). In patients, who were under 20 at the time of diabetes was discovered, the interval between this and visual loss was on an average 17.4 years (Fertz and Berkov 1968).

Above 20% of Juveniles with diabetes show changes in fundus (Chylinska and Abramowicz 1969; Darnaud et al 1963). The proliferative form appears in about 10% of cases of Juvenile diabetic retinopathy. Prognosis in these cases is poor, about 50% having less than 6/60 vision in both the eyes after 5 years (Deckert et al 1967). This type of Juvenile diabetic retinopathy appears between 10-14

years of age, whole simple or non proliferative type does not appear until 16-29 years (Michaelson 1980). The simple form of retinopathy appears in less than 10% of cases if diabetes has lasted for less than 10 years period; but it is found in atleast 70% after 20 years of diabetes (Knowles 1965). Kohner (1977) has suggested that there is an association between proliferative retinopathy and 'Juvenile onset' diabetes and between diabetic maculopathy and 'Maturity onset'. Bodanosky, Cudworth, Whitelock and Dobree (1982) also confirms this view. They also report association between male sex and proliferative retinopathy.

The reported incidence of retinopathy in Juvenile onset diabetes is greater than in adult onset group because the patients live long enough for the retinopathy to develop. The highest percentage, 80% occurred in cases of more than 15 years duration of diabetes. The onset of retinopathy in 'Juvenile diabetes' occurs after atleast 6-7 year (Borta and Molnar 1970). Terne (1972) reported retinopathy in 67% of cases of Juvenile onset diabetes compared to 43% of adult onset diabetes.

#### Neuro-ophthalmic association of diabetes :

Diabetes can be the cause of extraocular muscle palsies involving the 3rd (oculomotor), 4th trochlear and 6th abducens cranial nerves. The hallmark of diabetic nerve palsies is that they resolve the recovery or begning of recovery in a few weeks to 4-6 months. Any nerve palsy

which does not show signs of recovery in 6 months may well not be diabetic origin (Brude et al 1985).

The association of optic nerve hypoplasia in a child with a diabetic mother was first noted by Peterson and Walter (1977) and then elaborated by Nelson Ellessell and Sadum (1986). The quare~~det~~ of diabetes mellitus, diabetes insipidus optic atrophy and deafness is referred to Wolfram's syndrome (DIDMOAD) is commonly seen in juvenile diabetes. Lessell and Rosman (1977), Wolfram (1938). An increased occurrence of diabetes mellitus 20% has been noted in patients with anterior ischemic optic neuropathy of the non arteritic variety. Guyer (1985), Ellenberger (1973), Repka (1983).

Burde (1985) used term vasculopathic mononeuropathies involving 3rd nerve with pupillary sparing Rucker (1958), Goldstein (1960). Bell's palsy may precede a diabetic ophthalmoplegia in about one third of cases by several months to years Jaffe (1967).

Patients with diabetes mellitus are known to develop infections processes including orbital cellulitis characterized by proptosis, periorbital swelling and ophthalmoplegia Burde (1985), Rootman J and Lippincott, JB (1988).

SUBJECTS,  
METHOD AND  
MATERIAL



## MATERIAL AND METHODS

### MATERIAL :

The present study has been carried out in the department of Ophthalmology in active collaboration with diabetic clinic, department of Medicine, M.L.B. Medical College, Jhansi.

The selection of cases was done from the patients attending diabetic clinic and also diabetic patients attending the out patient department of Ophthalmology during their routine eye checkup. These patients belonged to Jhansi and near by district of U.P. and M.P. A total of 106 cases were selected for the study from 1995 to 1996.

### METHOD :

Details of the findings of each patient was recorded on the prescribed working proforma. General information of patient along with duration of disease, duration of treatment, type of treatment whether insulin or non-insulin dependent, control of blood sugar, any complications associated with diabetes was also enquired. Ocular history was obtained in detail and complete ophthalmic examination was done which include recording of the following :

1. Visual acuity :

This was recorded in every patient with the help of Snellen's distant chart (Snellen 1862), if vision was very much impaired then hand movements, perception of light and projection of rays was recorded.

2. Examination under difuse light :

By the help of well focussed ordinary torch eyes were examined externally under diffuse light. Orbit and face was examined for any facial asymmetry or deformity Eyebrows for any hair loss, movements of eye ball and position, eye lashes for partial or complete loss, regular or irregular pattern. Lid margins for any signs of inflammation, sty and drooping of lids and inrolling or outrolling of lid margins.

Conjunctiva was examined for conjunctival blood vessels any aneurysms, congestion, haemorrhage, conjunctivitis and nodule. Non specific changes such as pterygium, pinguecula and xerosis were also noted. Sclera for any signs of inflammation viz. scleritis and episcleritis and cilliary staphyloma. Cornea was examined for superficial interstitial and exposure keratitis, vascularisation of cornea, opacity and ulcer were also noted, if ulcer was suspected, it was confirmed by staining of cornea with 2% fluorescein solution, corneal sensation was also tested with



cotton wisp. Anterior chamber was examined for its depth and contents especially to find out keratic precipitate, flare.

Iris was examined for colour, surface, pattern any neovascularization, synechiae and atrophy.

Pupil was examined for size, shape and reaction to light. Lens was examined for any opacity or pigmentation over its anterior capsule.

#### EXAMINATION UNDER LOCAL ILLUMINATION :

Slit lamp examination of each case was done in a semidarkened room. In ophthalmic department. Patient was made to sit on stool and rest his head in a proper position. All the structures of anterior eye were examined by various methods of illumination, i.e. diffuse illumination. Sclerotic scatter, direct focal illumination, direct and indirect retroillumination, zones of specular reflection and indirect illumination.

Details of the lesions of lids, conjunctiva, cornea, anterior chamber, iris and lens were noted. Special attention was given to find out early punctate keratitis, staining of cornea, folds in decemets membrane.

4. Colour vision test was done in patients having visual acuity of 6/6 by ishihara colour plates. Patients having cataract and aphakia were not included.

5. Tonometry : Intraocular tension was recorded by standard certified indentation schiottz's tonometer under surface anaesthesia, 4% xylocaine instilled into eye.
6. Fundoscopy : Pupils of the patients eyes were dilated using 10% phenyl ephrine drops and fundus was seen by direct ophthalmoscope and the retinal status was graded into 4 grades.
  - a) Fundus with no significant findings.
  - b) Diabetic retinopathy stage I (Background retinopathy).
  - c) Diabetic retinopathy stage II (Preproliferative retinopathy).
  - d) Diabetic retinopathy stage III (proliferative retinopathy).

AIMS OF STUDY :

To study the ocular changes seen in diabetes mellitus in Bundelkhand region.

# WORKING PROFORMA

## STUDY OF OCULAR CHANGES IN DIABETES MELLITUS IN BUNDELKHAND REGION

Case No. :  
OPD / MRD No. :  
Date :  
Name :  
Age / Sex :  
Literate / Illiterate :  
Address :  
Socio Economic Status :  
Age at onset and duration of Diabetes :  
Type of Diabetes IDDM / NIDDM :  
Ocular History :

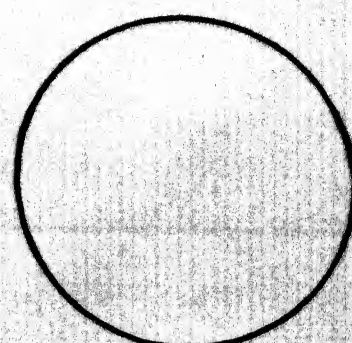
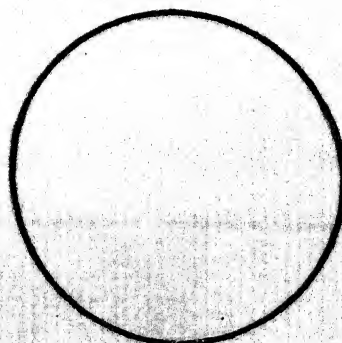
### Ocular Examination

Head Posture :  
Facial Symmetry :

RE

LE

Orbit :  
Eye Brows :  
Eye Ball Position and Movements :  
Eye Lids :  
Palpebral Aperture :  
Conjunctiva :  
Cornea :  
Sclera :  
Anterior Chamber :  
Iris :  
Pupil :  
Lens :  
Lacrimal Apparatus :  
Vision :  
Colour Vision :  
Tension :  
Field of Vision :  
Slit Lamp Examination :  
Fundus :



# OBSERVATIONS



### OBSERVATION

The present study was carried out in 106 Diabetic patients attending Diabetic Clinic, Department of Medicine as well as Department of Ophthalmology, M.L.B. Medical College, Jhansi over a period of one year from October, 1995 to October, 1996.

A total of 106 cases were examined out of them total number of males were 64 (60.3%) and total no. of females were 42 (32.62%) with a male to female ratio of approximately 3 : 1.

Table no. I : Distribution of patients according to age

Sl. No.	Age in years	No. of patients	Percentage
1.	Less than 20	4	3.77
2.	21 - 40	24	22.64
3.	41 - 60	56	52.83
4.	More than 61	22	20.75
Total		106	100.00



Table no. 1 shows age-wise distribution of diabetic patients. As is evident most of the 56 (52.83%) cases in the age group of 41-60 years. However, 24 (22.64%) cases were seen in the age group of 21-40 years and 22 (20.75%) cases were of 61 years and only 4 (3.77%) cases were below 20 years of age.

Table no. II : Sex-wise distribution of patients

Sl. No.	Age	Male	Female	Total
1.	0 - 20	4	0	4
2.	21 - 40	10	14	24
3.	41 - 60	36	20	56
4.	61 - above	14	8	22
Total		64 (60.38%)	42 (39.62%)	106

Table no. II shows sex-wise distribution of total cases according to age group. As can be seen from the table II, a total of 64 (60.38%) cases were among males, while 42 (39.62%) were seen among females. Males were more as compared to females in all age groups except in age group of 21 to 40 years. Females were 14 in number and males were just 10 in number.

Table no. III : Distribution of cases according to type of Diabetes mellitus

Sl. No.	Group	No. of cases	Male	Female	Percentage
1.	IDDM	28	14	14	26.42
2.	NIDDM	78	50	28	73.58

Table no. III shows type of Diabetes mellitus in the cases seen. Maximum 78 (73.58%) cases belonged to non-insulin dependent diabetes mellitus out of which 50 (64.10%) were males and 28 (35.89%) were females. 28 cases were of Insulin dependent diabetes mellitus, out of which 14 (50%) were males and 14 (50%) were females.

Table no. IV : Ocular involvement among different age group.

Sl. No.	Age in years	Total no. of cases	No. of cases ocular involvement
1.	Less than 20	4	4
2.	21 - 40	24	18
3.	41 - 60	56	52
4.	61 - above	22	21
Total		106	95

Table no. IV shows ocular involvement among different age groups. As is evident most of cases 56 (54.73%) were seen in patients above age of 40 years and ocular involvement was less 4 (4.21%) in age group less than 20 years.

Table no. V : Showing ocular manifestation with duration of diabetes.

Sl. No.	Duration of disease (Yrs)	No. of patients	Ocular involvement	Percentage
1.	0 - 5	20	4	20.0
2.	6 - 10	46	30	65.22
3.	11 - 15	16	12	75.00
4.	16 - 20	10	8	90.00
5.	21 - 25	10	10	100.00
6.	Above 25	4	4	100.00

Table no. V shows duration of disease. The ocular manifestation were of increasing trend as the duration of diabetes increases. It was nearly 100% in above 20 years of duration of diabetes and it was 20% in below 5 years duration of diabetes.



Table no. VI : Showing the involvement of ocular adnexa

Sl. No.	Lesions	No.of cases	Male	Female	Percentage Total no.of pts. - 106
1.	Facial muscle Palsy	-	-	-	-
2.	Trichiasis	4	-	4	3.77
3.	Blepharitis	8	3	5	7.54
4.	Recurrent-stye	11	4	7	10.37
5.	Ptosis	1	1	-	0.94
6.	Lagophthalmos	-	-	-	-
7.	Acute dacryocystitis	1	1	-	0.94
8.	Chronic dacryocystitis	1	-	1	0.94
9.	Orbital cellulitis	-	-	-	-
10.	Ophthalmoplegia	-	-	-	-

Table no. VI shows involvement of ocular adnexa in diabetic patients. The following observations were made.

Most common finding was Recurrent-stye which was present in 11 (10.37%) cases, (4 males and 7 females). Other most common finding was blepharitis, mostly ulcerative type in 8 (7.54%) cases out of which 3 males and 5 females. Trichiasis was seen in 4 (3.77%) cases all females.

Ptosis was seen in one patient (0.94%) which improved after 3 months. Acute dacryocystitis was seen in one male patient and chronic dacryocystitis in one female patient. Facial muscle palsy, lagophthalmos and orbital cellulitis was not seen among 106 cases, ophthalmoplegia was not seen in any case.



Table no. VII : Showing involvement in conjunctiva and Sclera in diabetes.

Sl. No.	Conj. lesions	Male	Female	Total	Percentage
1.	Conjunctivitis	1	2	3	2.83
2.	Xerosis	2	-	2	1.87
3.	Pterygium	0	1	1	0.94
4.	Pingicula	-	-	-	-
5.	Scleritis	-	-	-	-
6.	Episcleritis	-	-	-	-

Table no. VII shows involvement of conjunctiva. Conjunctivitis was present only in 3 (2.83%) cases and Xerosis in 2 (1.87%) cases and Pterygium in one (0.94%) case. No changes in Sclera were noted.

Table no. VIII : Showing the involvement of Cornea as seen on slit lamp examination.

(Total no. of patients - 106)

Sl. No.	Lesions	Male	Female	Total(%age)
1.	Superficial punctate keratitis	3	2	5 (4.71%)
2.	Exposure keratitis	-	-	-
3.	Corneal straiate	3	1	4 (3.77%)
4.	Decreased corneal sensitivity	12	3	15 (14.15%)
5.	Corneal opacity	3	2	5 (4.71%)
6.	Corneal ulcer	1	-	1 (0.94%)
7.	Anterior staphyloma	-	-	- -
Total		22	8	30 (28.28%)

Table no. VIII shows involvement of cornea in different ways in diabetic patients. The most common entity found was decreased corneal sensitivity which is in 15 (14.15%) cases and next common finding was superficial punctate keratitis and corneal opacity in 5 (4.7%) cases.

Other findings were less common such as corneal straiate in 4 (3.77%) cases and only one (0.94%) case of corneal ulcer.

There were no cases of Anterior staphyloma.

Table no. XI : Showing the involvement of anterior Uveal tract in Diabetic patients.

Sl. No.	Lesions	Male	Female	Total (%age)
1.	Acute iridocyclitis	3	1	4 (4.77%)
2.	Chronic iridocyclitis	-	-	+-
3.	Iris atrophy	-	2	2 (1.88%)
4.	Pigmentary changes	2	1	3 (2.83%)
5.	Rubeosis iridis	1	-	1 (0.94%)
6.	Fixed pupil	6	3	9 (8.49%)

Table no. IX shows involvement of anterior uveal tract in diabetic patients.

Fixed pupil not responding sluggish reacting to light was noted in 9 (8.49%) patients among them 6 were male and 3 females.

Acute iridocyclitis was present in 4 (3.77%) cases, out of them 3 were male and one female.

Pigmentary changes were noted in 3 (2.83%) cases, out of them 2 were male and one female.

Rubeosis iridis was seen in one (0.94%) patient who also had glaucoma.

Signs of iris atrophy were seen in 2 (1.88%) patients, which were females.

Table no. X : Showing lenticular changes in diabetes according to age.

Sl. No.	Age in years	Total cases/ Total eyes	No. of eyes with lenti- cular changes	Percentage
1.	0 - 2	4 (8)	8	100.00
2.	21 - 40	24 (48)	6	12.50
3.	41 - 60	56 (112)	42	37.50
4.	61 - above	22 (44)	18	40.90
Total		106 (212)	74	34.90

Table no. X shows incidence of lenticular changes in diabetes were about 34.90%.

Maximum no. of cases were of 42 (37.5%) in 41-60 years age group. Mostly of senile cataract type. True diabetic cataract was seen in cases below 20 years of age in 8 eyes.

In age group 61 and above, the incidence of cataract increased than other age groups about 40.9%. Cataract was common in all age groups as it is evident in above table.



Table no. XI : Distribution of diabetics patients having lenticular changes according to sex.

Sl. No.	Age	Total cases	No.of eye having cataract	<u>Total cases</u>		Percentage
				Male	Female	
1.	0 - 20	4	8	4	0	100.0
2.	21 - 40	24	6	2	1	12.5
3.	41 - 60	56	42	5	16	37.5
4.	Above 61	22	18	3	6	40.9
Total		106	74	14	23	

Table no. XI shows distribution of diabetic patients according to sex. Total no. of females were 23 and males 14. In age group 41 to 60 years, females were 16 and males were only 5.

In age group above 61 females out numbered males by 6 females and 3 males.

In age group less than 20 years, there were 4 cases of which all were males.

Table no. XII : Showing the visual acuity in total number of eyes and visual acuity in eyes with lenticular changes with best possible correction.

Sl. No.	Vision	Total no. of eyes	No. of eyes having lenticular changes	Other cause
1.	6/18 or better	116	10	106
2.	6/24 to 3/60	82	54	28
3.	Less than 3/60	14	10	4
4.	Total no. of eyes	212	74	138
5.	Percentage	100%	34.90%	65.0%

Table no. XII shows visual acuity in eyes with lenticular changes and eyes having any other cause attributing to diminution of vision like complication of diabetic retinopathy, corneal opacity, vitreous haemorrhage refractive error etc.

Blindness due to cataract was in 10 (4.71%) eyes and other causes in 4 (1.88%) eyes.

Vision was very much impaired 6/24 to 3/60 in eyes having lenticular changes in 54 (65.85%) eyes, while other causes in 28 (34.14%) eyes.

Table no. XIII : Range of intraocular tension in various grades of Retinopathy.

Sl. No.	Intraocular Tension	Normal fundus	Stage I	Stage II	Stage III
		No. of cases	No. of cases	No. of cases	No. of cases
1.	12.4 to 14.6 mmHg	-	-	-	12
2.	16.9 to 17.3 mmHg	25	17	10	14
3.	18.9 to 20.6 mmHg	15	32	27	-
4.	22.4 to 24.3 mmHg	-	9	11	-

Table no. XIII shows ocular tension in different grades of retinopathy. In total there were 86 cases (172 eyes) in which fundus was visible and intraocular tension was recorded in these patient only. Those patients having normal fundus had intraocular tension in normal range 16.9 to 20.6 mmHg. The patients belonging to stage I retinopathy had intraocular tension in 49 eyes was within normal (16.9 to 20.6 mmHg) range and 9 (15.5%) eyes having raised intraocular tension (ranging 22.4 to 24.3 mmHg).

The patients belonging to stage II retinopathy had intraocular tension in normal range in 37 (77.08%) eyes and raised intraocular tension in 11 (22.91%) eyes.

Stage III group had tension towards lower side in 12 (46.15%) eyes and normal intraocular tension in 14 (53.84%) eyes.

Table no. XIV : Showing Mean intraocular tension in relation to retinal status.

Sl. No.	Stages of diabetes	No. of eyes	Mean	S.D.	Range
1.	Diabetes with normal fundus	40	17.93	1.0	16.9 - 20.6
2.	Diabetes with retinopathy	132	18.95	2.38	12.4 - 24.3
	a) Diabetes stage-I	58	19.12	1.82	16.9 - 24.3
	b) Diabetes stage-II	48	19.95	1.91	16.9 - 24.3
	c) Diabetes stage-III	26	15.49	1.50	12.4 - 17.3

Table no. XIV shows mean intraocular tension in different grades of retinopathy.

Mean intraocular tension in normal fundus was 17.93 where as in diabetes with retinopathy, it was 18.95 towards higher side as compared to normal fundus.

Mean intraocular tension was maximum 19.95 in diabetes stage II retinopathy.

Mean intraocular tension was towards lower side 15.49 in stage III retinopathy as compared to diabetes with normal fundus.



Table no. XV : Showing percentage distribution of diabetic retinopathy status according to age of patient.

Sl. No.	Age in years	No diabetic retinopathy	Background diabetic retinopathy	Preproliferative diabetic retinopathy	Proliferative diabetic retinopathy	Total no. of cases
1.	21 - 40	12 (80.0%)	8 (40.0%)	-	-	20
2.	41 - 60	12 (22.22%)	15 (27.77%)	20 (37.03%)	7 (12.96%)	54
3.	61 - above	2 (11.11%)	6 (33.33%)	4 (22.22%)	6 (33.33%)	18

Table no. XV shows percentage distribution of diabetic retinopathy status according to age of patients. Age group below 20 years had diabetic cataract, so fundus was not visible.

In age group of 21-40 years 12 (60%) cases had normal fundus and 8 (40%) cases had background diabetic retinopathy.

In age group 41-60 years 12 (22.22%) cases had no diabetic retinopathy, 15 (22.77%) cases had background retinopathy, 20 (37.03%) cases had preproliferative retinopathy, and 7 (12.96%) cases had proliferative retinopathy.

In age group of 61 and above 2 (11.11%) cases had no diabetic retinopathy, 6 (33.33%) cases had background retinopathy, 4 (22.22%) cases had preproliferative retinopathy and 6 (33.33%) cases had proliferative retinopathy.

In age group 21 to 40 years, there were less chances of developing preproliferative diabetic retinopathy and proliferative retinopathy. Chances of preproliferative retinopathy and proliferative retinopathy were more in higher age groups.

Table no. XVI : Showing sex distribution by duration of diabetes and retinopathy status.

Sl. No.	Duration of Diabetes (Years)	NDR		BDR		PPDR		PDR		TOTAL	
		Male	Fe- male	Male	Fe- male	Male	Fe- male	Male	Female	Male	Female
1.	0 - 5	12	8	-	-	-	-	-	-	12	8
2.	6 - 10	4	2	12	15	2	1	-	-	18	18
3.	11 - 15	-	-	1	1	4	6	-	-	5	7
4.	16 - 20	-	-	-	-	5	2	2	1	7	3
5.	21 - 25	-	-	-	-	3	1	5	1	8	2
6.	26 or more	-	-	-	-	-	-	2	2	2	2

Table no. XVI shows sex distribution by duration of diabetes and retinopathy status.

No. of male were more in NDR group than female.

No. of females were more in BDR group than males.

No. of males were more in PPDR and PDR group than females.

In total there were 30 (71.42%) females having retinopathy out of 42 and 36 (56.25%) males having retinopathy, out of 64 males.



**Table no. XVII : Showing pattern of diabetic retinopathy according to duration of diabetes.**

Sl. No.	Duration	Total cases	Normal	Stage-I	Stage-II	Stage-III
1.	Upto 5 yrs.	20	20 (100%)	-	-	-
2.	6 - 10	36	6 (16.66%)	27 (75.0%)	3 (8.33%)	-
3.	11 - 15	12	-	2 (16.6%)	10 (83.33%)	-
4.	16 - 20	10	-	-	7 (70.0%)	3 (30.0%)
5.	21 - 25	10	-	-	4 (40.0%)	6 (60.0%)
6.	Above 25	4	-	-	-	4 (100%)

Table no. XVII shows pattern of diabetic retinopathy according to duration of diabetes.

Duration of diabetes below 5 years had no diabetic retinopathy.

Duration of diabetes 6 to 10 years had mostly stage-I diabetic retinopathy (75%).

Duration of diabetes 11 to 15 years had mostly stage-II diabetic retinopathy (83.33%).

Duration of diabetes 16 to 20 years and 21 to 25 years had both stage-II and Stage-III diabetic retinopathy.

Duration of diabetes more than 25 years had stage-III diabetic retinopathy.

--- --

## DISCUSSION

### DISCUSSION

Diabetes mellitus is characterized by sustained hyperglycemia secondary to lack or diminished efficacy of endogenous insulin and is one of the most common diseases in the civilized world.

Each part of visual system is susceptible to the harmful effects of diabetes which range from changing refractive errors to progressive disease of eye itself including cornea, iris, lens, retina optic nerve, extraocular muscles and orbit.

As per review of literature it will be noted that although ocular complications in diabetes are rated high in most of the western countries. Yet there are considerable variations in its incidence as reported by different workers all over the world. In India, very little has been reported on this subject in the past few decades the reason for this appears to the lack of regular ophthalmological survey of diabetic patients.

The cases, were selected randomly from diabetic clinic of Department of medicine and from Ophthalmology department, M.L.B. Medical College, Jhansi.

The present study includes 106 cases, which are mostly males 64 (60.37%) and rest females 42 (39.62%), these may be due to more prevelage given to man.



over women in Bundelkhand region in every field from education to even sickness and awareness of Health. Maximum number of patients were from the age group of 41-60 years (52.89%) followed by 22.64% in age group of 21-40 years and 20.75% in 61 and above age groups.

Most of the patients belonged to urban areas, which may be due to less general awareness and ignorance among rural people of Bundelkhand region.

Diabetic patients were divided into two major groups according to type of diabetes mellitus. Type-I insulin dependent diabetes or Juvenile onset diabetes which comprised of 28 (26.42%) cases and Type-II non-insulin dependent diabetes or maturity onset diabetes which comprised of 78 (73.58%) cases. The prevalence of Type-I diabetes in Scandinavia was 20% and in Southern Europe 13%, 8% in the USA according to reports of John (1994).

Type-II diabetes (NIDDM) comprises large group as reported 75-80% of North Americans and Europeans.

Regarding the age incidence, the age group of the patients varied from 7 years to 72 years, maximum no. of cases belonged to 4th decade to 6th decade. Sosby (1966) also noted maximum number of cases which belonged to 4th to 6th decade.

Out of total 106 cases in our study, ocular involvement were noted in 95 (89.62%) cases incidence of ocular involvement studied by Kisby (1967) came out to be 64% of all diabetics, who had lenticular changes of same kind or other, ocular symptoms occur in between 20% to 40% of diabetics at the clinical onset of the disease Lister (1935), refractive changes upto 47% Granstrom (1933).

In the present study it was seen ocular involvement was more common in older age groups, in table no. IX, 52 cases out of 56 having ocular involvement in one way or other in age group 41-60 years, and 21 cases out of 22 in age group above 61 years.

Ocular involvement was more in patients having longer duration of diabetes. With duration of diabetes less than 5 years had 20% involvement, and those with 6-10 years 65.22% and 11-15 years 75% and 16-20 years 90% above 21 years had nearly 100% involvement in one way or other.

Involvement of ocular adnexia was in 26 cases. The following lesions were noted facial muscle weakness, no cases were seen.

Trichiasis was observed in 4 (3.78%) cases which comprised of females only. Blepharitis was observed in

8 (7.54%) cases, Recurrent Sty was observed in 11 (10.37%) cases. A. Dolenek and A. Takac (1967) started the involvement of lids in these conditions but about the incidence no data is available.

Acute dacryocystitis was seen in one (0.94%) case and chronic dacryocystitis in 1 case.

Ptosis was observed in one case. There was no case of ophthalmoplegia, although oculomotor nerve palsies have been reported by Brude et al 1985, Rucker 1958, Goldstein 1960, Jaffe 1967. Some of workers have reported that ophthalmoplegia is not common. Waite & Beetham 1935 found a frequency of only 0.4% in 2000 cases of diabetes.

There was no case who had orbital cellulitis, which was noted by Burde (1985), Rootman and Lippincett (1988). The main reason may be the number of cases seen in this study is very less than the study carried by them.

Involvement of conjunctiva was seen in 6 (5.64%). In 3 cases conjunctivitis was seen and 2 cases of Xerosis and 1 case of pterygium, pingicula, scleritis and episcleritis was not seen in any cases.

Corneal lesion in diabetic patients were found in 30 (28.28%) cases. Decreased corneal sensitivity was found markedly impaired in 15 (14.15%) cases. These

findings as compared with Prakash and Kannan (1993) reported 42% of cases of diabetes having decreased corneal sensitivity.

Superficial punctate keratitis was seen in 5 (4.71%) cases and corneal opacity was seen in 5 (4.71%) cases, corneal straiate was seen in 4 (3.77%) cases. One case had corneal ulcer (0.94%). Exposure keratitis and anterior staphyloma was not seen in any case.

Involvement of iris was observed in 9 (8.49%). Acute iridocyclitis was seen in 4 (3.77%) cases. Studies conducted by Marchal (1863) have reported involvement of iris as iritis or iridocyclitis in 0.8 to 8% of cases.

Waite and Beetham (1935) reported about 1.3% of iritis.

Pigmentary changes was seen in 3 (2.83%) cases in our study. Becker (1883), Armaly and Baloglon (1967), Waite & Beetham (1935) in their study found out pigment deposits in various sites in the anterior chamber in almost a third of diabetics, on anterior surface of iris and posterior surface of cornea in 10% of cases and no lens in 3% of cases.

Iris atrophy was observed in 2 (1.88%) cases, these were above 70 years age group patients.



Rubeosis iridis was observed in only one (0.94%) case in our study. A figure of less than 1% is much more reasonable than Janert et al (1957) but according to Alagna & Scullica (1956). Ohrt (1958) the frequency of rubeosis iridis was about 5%.

Fixed pupillary reflex was observed in 9 (8.49%) cases. Involvement of pupil in diabetic patients studied by another study was around 10% in patients, Rucker (1958), Goldstein and Cogan (1960).

Lenticular changes were observed in 74 (34.90%) cases, our result is nearly same as reported earlier by Janert (1960), who found lenticular changes in 40% of cases and 5% of cases belonged to age group upto 16 years.

Cataract was common in all age group in our study. Flanagan D.W. (1960) also noted that cataract was common in all age groups. Maximum number of cases were of 41-60 years age group. The age at which cataract surgery was performed in diabetic patients was significantly earlier.

Lenticular opacities were more common in female diabetics than the male diabetics. There were 23 females and 14 males. This result is comparable to the report of Heinsius and Arndt 1950, Janert et al 1956.

Blindness due to cataract in diabetic patients was in 10 (4.77%) eyes. This result when compared to another study in West of Scotland (1910) suggested about 25.33% blindness due to cataract among diabetics is higher than our study.

Visual acuity of 6/18 or better was noted in 116 eyes and among these 10 eyes had lenticular changes.

Visual acuity of 6/24 to 3/60 was noted in 82 cases and among these 54 eyes had lenticular changes.

Visual acuity of less than 3/60 was noted in 14 eyes and among these 10 cases had lenticular changes.

#### INTRAOCULAR TENSION :

Intraocular tension was recorded in selective patients in which fundus was visible to assess the changes related to retinopathy. There were 26 cases (172 eyes) in which fundus was visible and intraocular pressure was recorded. The range of intraocular pressure varied in all the three grades of retinopathy as well as in patients having fundus findings within normal limits.

Range of intraocular tension varied in various grade of retinopathy, it was towards lower side in Grade III 12.4 to 14.6 mmHg.

Higher side in Grade I and Grade II, ranging from 16.9 to 24.3 mmHg.

Patients having no retinopathy had intraocular tension within normal range of 16.9 to 20.6 mmHg.

It is evident from table no. 14, that total of 40 (23.25%) eyes, of diabetics had no retinopathic changes in fundus and mean intraocular pressure in them is  $17.93 \pm 1.0$  which is within normal range of 16.9 to 20.6 mmHg.

Another important interesting observation of our study was that, as the grade of retinopathy increased, the mean intraocular pressure also showed an increasing tendency until at the final stage grade III retinopathy it declined steeply.

In grade I retinopathy, out of 58 (33.72%) eyes mean average intraocular pressure is found to be  $19.12 \pm 1.82$  with range of 16.9 to 24.3 mmHg, we find a definite increase in the mean intraocular pressure as when compared to patients having no retinopathic changes in fundus.

Grade II retinopathy, out of 49 (27.90%) eyes, similarly, following the tradition showed an increase in mean average intraocular pressure i.e.  $19.95 \pm 1.91$  and ultimately in grade III retinopathy out of 26 eyes which

formed a total of 15.11%, showed  $15.43 \pm 1.50$  of mean intraocular pressure which when compared to those having no retinopathy in fundus  $17.93 \pm 1.0$  shows quite good amount of difference.

On comparison of mean intraocular pressure observed by us in different grade of retinopathies to that of other workers, we find a similar correlation as found by Arora and Prasad (1983). They found mean intraocular pressure in diabetics without retinopathy to be 18.17 mmHg while in eyes with retinopathy, it was 19.99 mmHg (In our study the intraocular pressure in diabetics with retinopathy is  $18.95 \pm 2.38$  and in diabetics without retinopathy  $17.93 \pm 1.0$ ). Arora and Prasad found significant difference in mean intraocular pressure in proliferative retinopathy when compared with those having no retinopathy. In grade I retinopathy the mean intraocular pressure was 20.98 mmHg. in grade II 21.99 mmHg and proliferative retinopathy group had 15.22 mmHg.

In contrast to other findings, Christiansons (1960) who studied total of 172 diabetic patients, he found a decreasing schiotz tension as the grade of retinopathy increased viz 16 mmHg in grade I retinopathy in grade II, the tension decreased to 12.3 mmHg and grade III 9.3 mmHg.



RETINAL STATUS OF EYES :

Table no. 15 shows the retinal status of case material, cases with retinopathy which forms total of 66 i.e. 62.26% of the total case material is nearly close agreement with the prior studies viz. Wagener's (1921) found the incidence of 8.3% of diabetic retinopathy among diabetics (Wagener and Wilder 1921), in 1934, the incidence has risen to 17.7% (Wagener et al 1934) and in 1945, it was 29.6%, Komuruf (1957) found 601 cases of diabetic retinopathy out of 1285 unselected diabetics 47%, and Dollfus (1954) reported 52.4% cases. It, therefore, follows that the incidence of diabetic retinopathy is on an increase and is expected to have above 50% cases of diabetic retinopathy at present.

As has been broadly classified earlier table no. XV, the majority of cases 36 (54.54%) were males and 30 (45.45%) were females.

Retinopathy changes observed among the males 69.23% and among females, it was higher than males being 75%. Duke Elder describes that female - male ratio is 3 : 2 and also that women are more liable to develop retinopathy. In other studies there is much greater incidence among females. Hanum (1938) for eg. in 183 cases of diabetic retinopathy found 72% females and 28% males. The larger statistics of

Portsmann and Wiese (1954), Keiding et al (1952), Janert et al (1956) and Babel and Rilliet (1958), however, did not show a difference between the sexes. We also observed that retinopathy is more common in patients of age group above 40 years or advance age and rare in below 40 years. These findings are very much in correlation with studies by Forsyth and Paynt 1956, Imorlund (1959), Girner 1960, Guest Lample Kessler and Skillman 1965.

As has been broadly classified earlier table no. XV amongst the different grades of retinopathy, there were 16 males and 10 females (28.26%), in grade 0 retinopathy.

In grade I retinopathy - there were 13 males and 16 females (31.53%).

In grade II retinopathy - there were 14 males and 10 females (26.08%).

In grade III retinopathy - there were 9 males and 3 females (13.04%).

In various grade of retinopathy - study according to Christionsons (1960) found maximum number of cases in grade 0 viz. 33 males and 49 females. 17 males and 19 females in grade I, in grade II, 6 males and 6 females, in grade III 7 males and 4 females and finally 11 males and 8 females in grade IV showing decreasing order.

In our study we observed that the diabetic retinopathy develops in diabetic patients in duration of 16 to 20 years to have all grades of retinopathy while Christianssons (1961) reported retinopathy to develop on an average 16.7 years of duration. In our study we found duration of diabetics more than 5 years is required to develop any retinopathy a figure which is close relation with the studies of Kornurup (1955) upstem workers in the past had reported that a minimum of 3 years period is required to develop any retinopathy (Waite and Beethaman 1935, Wagener 1945, Friend-enwald 1950, Lawrance et al 1951, Gardes 1953, Scott (1953).

To develop proliferative retinopathy, our study showed an average duration of 16.5 years on the other hand, patients with proliferative retinopathy constitute 17.5% of total case material with average duration of diabetes of 16 years. According to study by Christianssons (1960) and Kornurup (1958) also had same figures of 16.1 year and diabetic duration to develop proliferative retinopathy.

In our study with relation to duration of diabetes, table no. XVII, it was observed that with duration of diabetes upto 5 years had no retinopathy.

With duration of diabetes upto 10 years 16.66% had no retinopathy, 75% had grade I retinopathy and 8.33% had grade II retinopathy.

With duration of diabetes upto 15 years had 16.60% cases with grade I retinopathy and 83.83% had grade II retinopathy.

With duration of diabetes upto 20 years 70% had grade II retinopathy and 30% had grade III retinopathy.

With duration of diabetes upto 25 years 40% had grade II retinopathy and 50% had grade III retinopathy.

With duration of diabetes more than 25 years had only grade III retinopathy.

These findings clearly suggests that with duration of diabetes, the severity of diabetic retinopathy also increases. In another study by Khan HA and Broadly RE (1975), the prevalence of diabetic retinopathy among patients at Joslin Clinic was 25% in total diabetic population, 7% with less than 10 years duration, 26% with 10 to 14 years duration, 63% in patients with 15 years or more duration of diabetics. Dollfun and Haige (1953) 90% in patients of over 18 years duration of diabetics. Same findings were observed by Khosla et al (1984).

In Nutshell, our study amply demonstrates and collaborates the earlier findings, that diabetes affects all the structures of eye in one way or other, including



lenticular changes, mean intraocular pressure increases as the grade of retinopathy increases ultimately showing a downfall at proliferative retinopathy stage.

The study has also indicated that as the duration of diabetes is increased, it is directly related to the retinopathic grade.

---.---.---

## CONCLUSION

### C O N C L U S I O N

Diabetes mellitus is characterized by sustained hyperglycemia secondary to lack or diminished efficacy of endogenous insulin. Each part of visual system is susceptible to harmful effects of diabetes. Present study was conducted to see the ocular manifestations of diabetes mellitus, in Bundelkhand region.

In the present study 106 diabetics were examined in which 64 (60.37%) cases were males and 42 (39.62%) were females.

Maximum number of patients were from the age group of 41-60 years (52.83%) followed by 21-40 years (22.64%) and above 60 years (20.75%).

Type - I IDDM comprised of (26.42%) cases and type - II NIDDM (73.58%).

Incidence of ocular involvement among 106 diabetics was 89.62% and was related to duration of diabetes, rather than with the age of patients. With duration of less than 5 years 20% involvement and 6-11 years (65.22%) and 11-15 years 75% and 16-20 years 90% and above 21 years had nearly 100% involvement in one way or other.

Involvement of ocular adnexia was in 26 cases, Blepharitis was observed in 8 cases, Recurrent sty in 11 cases, Trichiasis in 4 cases, and one case each in Ptosis, Acute and Chronic dacryocystitis.

Non specific findings related to conjunctiva were conjunctivitis in 3 cases, xerosis in 2 cases and pterygium in 1 case.

Cornea got involved in 30 cases i.e. decreased corneal sensitivity in 15 cases, which is significant finding among other changes seen in cornea. Superficial punctate keratitis and corneal opacity in 5 cases, corneal striae in 4 cases and one case of corneal ulcer.

Iris was involved in 19 cases, acute iridocyclitis in 4 cases, pigmentary changes in 3 cases, iris atrophy in 2 cases, rubeosis iridis in one case. Pupillary reaction to light was found sluggish or fixed in 9 cases.

Lenticular changes were more common in females out of total 74 cases there were 23 females and 14 males. Another interesting finding was that the lenticular changes were seen in all the age groups, and it appeared earlier in diabetics and was an important factor for visual impairment.



Intraocular pressure was recorded in 86 cases in which fundus was visible. It was then compared in different grades of retinopathy. The mean intraocular tension in eyes without retinopathy  $17.93 \pm 1.0$  mmHg. Mean intraocular tension in eyes with retinopathy was  $18.95 \pm 2.38$  mmHg, this when compared to the eyes without retinopathy was towards higher side. Mean intraocular tension in grade I retinopathy was  $19.12 \pm 1.82$ ; in grade II  $19.95 \pm 1.91$ ; and finally in grade III, the mean intraocular tension  $15.49 \pm 1.50$  showed lower value than other grades of retinopathy including eyes without retinopathy.

Apart from all these changes we also studied the relation between the development of retinopathy and duration of disease leaving aside the age of the patient, and the severity of disease. The retinopathy seems to develop of anygrade on an average of 16.7 years of duration.

With duration of diabetes less than 5 years there was no retinopathy, with duration of diabetes upto 10 years 16.66% cases had no retinopathy 75% had grade I retinopathy and 8.33% cases had grade II retinopathy and none had grade III retinopathy.

With duration of diabetes, upto 15 years 16.6% cases had grade I retinopathy, 83.33% grade II and none had grade III retinopathy.

With duration of diabetes upto 20 years none had grade I retinopathy, 70% cases had grade II and 30% cases had grade III retinopathy.

With duration of diabetes upto 25 years none had grade I retinopathy, 60% cases had grade II and 40% cases had grade III retinopathy.

With duration of diabetes more than 25 years all cases had only grade III retinopathy.

These findings clearly suggests that as the duration of diabetes increases, the severity of diabetic retinopathy also increases.

---.---

# BIBLIOGRAPHY

### BIBLIOGRAPHY

1. Abrahamson JA, Jr, Abrahamson IS Sr. : Applanation and Schiotz tonometry. Am J Ophthal 1959; 48 : 389.
2. Alimuddin M. Normal intraocular pressure. Fr J Ophthalmol 1956; 40 : 371.
3. Armstrong JR, Dany RK, Dobson II, L, Giard LJ. The incidence of glaucoma in diabetes mellitus. Am J Ophthalmol, 1960; 50 : 55-63.
4. Arora VK, Prasad VN. The intraocular pressure and diabetes : A correlation study. Ind J Ophthalmol 1989; 37 : 1.
5. Ashton N. Vascular basement membrane changes in diabetic retinopathy. Br J Ophthalmol 1974; 58 : 349.
6. Backer Shaffers. Diagnosis and therapy of glaucoma, pp 573-82.
7. Babel J, Rilleit B : Diabetic retinopathy. Ophthalmologica (Basel, 1958; 135 : 5-6, 471).
8. Ballantyne AJ, Michadson IC. Text Book of the fundus of the retina. E and S Livingstone Edin and London, 1970, 221.
9. Ballantyne AJ, Lowenstein A : Diseases of retina I. pathology of diabetic retinopathy. Trans Ophthalmol Soc UK. 1943; 63 : 95.



10. Baste L, Molnar M. The data of appearance of diabetic retinopathy in children. *Holv Paeds Acta* 1970; 25 :242.
11. Bengt Jerneld and Deep Algvere. Relationship of duration and onset of diabetes to prevalence of diabetic retinopathy. *Am J Ophthalmol* 1986; 102 : 431-37.
12. Ben Erra A. The role of prostaglandin in the induction of neovascularization, 1978.
13. Beetham WP. Visual prognosis of proliferative diabetic retina. *Brit J Ophthalmol*, 1963; 47 : 6111.
14. Bernard Becker. Diabetes mellitus and primary open angle glaucoma. *Am J Ophthalmol* 1971; 71 (1); 1-16.
15. Bloodworth JMB Jr. Diabetic retinopathy, 1961; 11 : 1.
16. Bdoansky HJ, Cudwoeth AG, Whiteloche RAF, Bobrac JH. Diabetic retinopathy and its relation to type of diabetes. Review of a retinal clinic population. *Br. J Ophthal*, 1982; 66 : 496.
17. Braun R. Retinitis diabetica. *Arch Ophthalmol*, 136.
18. Burditt and Card FJ. *Brit J Ophthalmol*, 1968; 52 : 433.
19. Caird FI. Diabetes and the eye. Oxford Blackwell Scientific Publications 1969; 1-7.
20. Caird JI, Hutchinson and Piric. *Br Med J*, 1964; J2, 665-8.
21. Chatterjee A, Milton RC. *Br J Ophthalmol*, 1982; 66 : 35-42.

22. Cogan DG. Toussaint MD and Kuwabara T. Retinal vascular patterns. IV Arch Ophthalmol, 1961; 66 : 366.
23. Christiansson J. Intraocular pressure in diabetes mellitus. Acta Ophthalmol 1961; 39 : 159.
24. Cullen JF, Harris P, Turner JW and Duncan LJP, 1965. Pituitary stalk section for advancing diabetic retinopathy. Brit J Ophthalmol, 1965; 49 : 393.
25. Damato FJ. Brit J Ophthalmol 1960; 44 : 164-71.
26. Darnad C, Denard Y, Morean G, Voisin R. Frequencies of retinopathies in infantile diabetes of long duration diabetes. L C Rainey, 1963; 11 : 235.
27. Davis MD. Randomised controlled clinical trial National Eye Institute workshop for ophthalmologists Application of the principles of clinical trials. Am J Ophthalmol 1975; 79 : 779.
28. De Hase EBH. Desiccation of cornea and canj after sensory denervation. Arch Ophthalmol 1962; 67 : 79-92.
29. Diabetic retinopathy study research group. Preliminary report on effects of photocoagulation therapy. Am J Ophthalmol 1976; 81 : 383.
30. Dollfus MA. Acta Ophthalmol Canada, 1984; 1 : 119-27.
31. Dugmore WN Tunk. Glucose tolerance test in 200 patients with senile cataract. Br J Ophthalmol 1980; 64 : 689-92.
32. Duke Elders. Lancet, 1926; 1 : 1188 and 1250.

33. Duke Elders. Diseases of lens and vitreous. System of ophthalmology Vol 11 : 166-71, S. Duke Elders et St Louis CV Mosby Col 1969.
34. D.W. Flanagan diabetes glaucoma sex and cataract. Brit J Ophthalmol 1993; 77 : 1.
35. Ederer F, Hiller R, Taylor HR. Am J Ophthalmol 1981; 91 : 381-395.
36. Engerman R. Blood-worth JMB, JR Nelson S. Relationship of microvascular diseases in diabetes to metabolic control. Diabetes 1977; 26 : 760.
37. Engerman RL. Animal models of diabetic retinopathy. Trans Am Acad Ophthalmol Otolaryngeal, 1976; 81 : 710.
38. Ederer F et al. Am J Ophthalmol 1981; 92 : 135-6.
39. Esmann V, Lundback K, Madsen PH. Type of in diabetic retinopathy. Acta Med Scan 1963; 174 : 375.
40. Fiascher JP. In modern trends in ophthalmology (2nd series) (Sorbsy A ed.), p 54-70; Butterworth London.
41. Friedenwald JS. Diabetic retinopathy. Am J Ophthalmol 1950; 33 : 1187.
42. Friedenwald JS. Diabetic retinopathy. Am J Ophthalmol 1954; 37 : 953.
43. Gobbay KM. N Eng J Med 1973; 288 : 831.



44. Gordon. Arch Ophthalmol 1949; 41 : 362.
45. Goulden. Trans Ophthalmol 1928; 48 : 97.
46. Halevi HS and Lander J. Br J Ophthalmol 1962; 46 : 285-90.
47. Hanum S. Diabetic retinitis, Clinical studies of 195 cases of retinal changes in diabetes. Acta Ophthalmol (Kbh) Suppl, 1938; 16 .
48. Harrison's Principles of Internal Medicine. 12th edition, Vol 2, 319; 1739-58.
49. Heinsius and Arndt et al. Arch Ophthalmol 1950; 158 : 555-71.
50. Hiller R, Khan HA. Br J Ophthalmol 1976; 60 : 283.
51. Hoffer KJ. Am J Ophthalmol, 1980; 90 : 260-8.
52. Huggert. Acta Ophthalmol (Kbn), 1954; 31 : 375.
53. Hutchinson M, Antoinette. Br Med J, 1962; 2 : 665-8.
54. IJ Constable et al. Assessing the risk of diabetic retinopathy. Am J Ophthalmol, 1984; 87 : 53-61.
55. Jack, Kanski. Clinical Ophthalmology, 3rd edition 1994; p 344-357-
56. Jackson. Br. J Ophthalmol 1955; 39 : 629.
57. Jackson CRS. Schiøtz tonometers. Br J Ophthalmol 1965; 49 : 478.



58. Jain IS, Luthra CL. Diabetic retinopathy. Its relationship with intraocular pressure.
59. John J Harding et al. Diabetes, glaucoma, sex and cataract. Br J Ophthalmol, 1993; 77 : 2-6.
60. Kahn HA, Bradley RF. Prevalence of diabetic retinopathy. Age, sex and duration of diabetes. Br J Ophthalmol, 1975; 59 : 345.
61. Kahn HA, Leibowitz HE et al. Am J Ophthalmol, 1977; 106 : 17-32.
62. Kahn HA, Kini MM, Calton T, et al. Am J Ophthalmol 1978; 85 : 28.
63. Kahn HA, Moorland HB. Statistics on blindness in the model reporting area. 1969-70, Publication No. 72,427.
64. Kamle Mala and Bhartiya SS. Ocular change in juvenile diabetics. Indian Ophthalmology to day, 1994 : p 256-258.
65. King RC, Dobree JH, Kok DA et al. Exudative diabetic retinopathy. Br J Ophthalmol 1963; 47 : 666.
66. Kirby. Arch Ophthalmol, 1933; 9 : 966.
67. Kirby, Esley and Wiener. Arch Ophthalmol, 1933 : 20 : 37.
68. Kirmani. Am J Ophthalmol 1964; 57 : 617.
69. Kornurup T. Studies in diabetic retinopathy. Acta Med Scandinal 1957; 153 : 81.

70. Krause. Am J Ophthalmol, 1934 ; 17 : 502.
71. Lawrence. Br J Ophthalmol 1946; 30 : 78.
72. Leot, Chylack JR et al. Loss of contrast sensitivity in diabetic patients with loss II classified cataracts. Br J Ophthalmol, 1993; 73 : 7-11.
73. Lloyd M. Alielle et al. The eyes and diabetes. Joslin's diabetes mellitus 12th edition, 1958; 600-34.
74. Marr WB. Am J Ophthalmol, 1952; 35 : 577.
75. McBrain EH. Arch Ophthalmol 1957; 57 : 520, and Arch Ophthalmol 1958; 60 : 1080.
76. Mooney AJ. Diabetic retinopathy: A challenge. Br J Ophthalmol 1963; 47 : 513.
77. Monique S Roy et al. Colour vision defects in early diabetic retinopathy. Arch Ophthalmol, 1968; 104 : 225-228.
78. Muh-Shy-chen et al. Prevalence and risk factors of diabetic retinopathy among non-insulin dependent diabetic subjects. Am J Ophthalmol, 1992; 114 : 723-30.
79. Newberg, Grisum, Burns. Diabetes, 1958; 7 : 21.
80. Norn. Acta Ophthalmol (Kbn), 1967; 45 : 322.
81. O'Brsans CS and Allen JH. J Am Med Assoc, 1942; 120 : 130-2.

82. Owens WC, Hughes WF. Arch Ophthalmol, 1947; 35 : 561-71.
83. Pahwa JM. Applanation tonometer. Prec of All India Ophthalmol Soc, 1960-61, 64.
84. Patterson JW. Am J Ophthalmol, 1952; 35 : 68.
85. Payman principles and practice of ophthalmol, 1987 Edition Vol 2, 1205-1276.
86. Perkins ES. Br J Ophthalmol, 1984; 68 : 293-7.
87. Pope CH. Retinal capillary microaneurysm. Diabetes, 1955; 4 : 24.
88. Pirie A. The human lens. In relation to cataract. Amsterdam Elsevier Publication, CIBA foundation Symposium, 1973; p 305.
89. Ramsell TE. Acute Diabetic Retinopathy, 1970; 7 : 789-99.
90. Robert MG. Br Med J, 1967; 2 : 416-18.
91. Robert a Hyndiuk et al. Neurotropic corneal ulcers in diabetes mellitus. Arch Ophthalmol, Dec, 1977; 95 : 2193-2196.
92. Ray Taylor et al. Comparison of non mydriatic retinal photography with ophthalmoscopy. Br Med J, 1990; 3011: 1243-1247.
93. Schwartz De. Corneal sensitivity in diabetics. Arch Ophthalmol, 1974; 91 : 174-178.



94. Schwartz JT, Delloze CG, Goldmann and Schiotz tonometry. Arch Ophthalmol 1966; 75 : 788.
95. Schiotz 1909, cited in Alimuddin M. Normal intraocular pressure. Br J Ophthalmol 1956; 40 : 371.
96. Scott GI. Discussion on diabetic retinopathy. Broc Roy Soc Med, 1957; 44 : 743.
97. Scott GI, Ocular complications of diabetes mellitus. Br. J Ophthal, 1953; 37 : 705.
98. Scott GJ. Ocular aspects of diabetes. Trans Ophthalmol Soc, V.K., 1957; 77 : 115.
99. SFJ Pilley. Familial syndrome of Didmoad in childhood. Brit J Ophthalmol 1976; 60 : 294-298.
100. Simm K. Arch Ophthalmol, 1962; 67 : 312.
101. Simmons D, William Dr Powell MJ. Prevalence of diabetes in a predominantly Asian community. B Med J, 1989; 298 : 18-21.
102. Skalka HW. Brenal JT. Ophthalmology, 1981; 86 : 117-24.
103. Smith JL, Bussey JL, Clack SW, Curtin VT et al. Arch Ophthalmol, 1967; 77 : 305.
104. Sommer A. Am J Ophthalmol, 1981; 92 : 134-5.
105. Sorbsy A. Reports on public health and medical subjects. 1966; 114 : 1948-62.



106. Spirdute RO, Seigeld Robert J. Rawland M. Arch Ophthalmol, 1982; 101 : 405-7.
107. Tannahil. Br Med J, 1885; 1 : 226.
108. Tulloch JA. (1962). Diabetes mellitus in the tropics. Ch 5 Livingstone, Edinberg.
109. Turtz C. Am J Ophthalmol, 1958; 46 : 219.
110. Verre HM (1969). Diabetes source book US. Department of health education and welfare, Public Health Service Publication, No. 1158.
111. Vere. Lancet, 1952; 2 : 1017.
112. Waite JH, Beetham WP. The visual mechanism in diabetes mellitus. Comparative study of 2002 diabetics and 457 non diabetics for control. N Eng J Med 1953; 212 : 367 and 429.
113. Waite and Beetham. New Engl J Med, 1935; 212 : 362-79.
114. Waite JH. Am J Ophthalmol, 1936; 7 : 124-36.
115. Wagner HE. Retinopathy in diabetes mellitus. Proc Amer Diab Assoc, 1945; 51 : 201.
116. Walker IB, Brown PE. Lancet, 1964; 51 : 201.
117. Walker JR. Diabetic retinopathy. Am J Ophthalmol 1961; 51 : 1123.
118. Wheeler. Trans Am Ophthalmol Soc, 1916; 14 : 742.

119. Wolfman HW et al. Cornea and diabetes. Arch Ophthalmol, 1929; 44 : 576-603.
120. Working party. Br Med J, 1962; 1 : 1497-1503 and 1963; 2 : 655-9.
121. Wright Barry E. Erra E, Kritzirger. Atlas of the eye and systemic diseases.
122. Yanoff M, Fine BS. Ocular pathology. A text and atlas chapter to nagerstorin MO Harper and Row p 566-85, 1975.
123. Yheberg J. Acta Ophthalmol, 1960; 38 : 548.

.....

# SUMMARY

### S U M M A R Y

Diabetes mellitus is the most common endocrine disease and the complications are known to occur in almost every part of visual organ some of these changes are of no importance and are not characteristics, other, however, are pathognomonic for diabetes. These changes are related to duration of diabetes, age of onset and control of diabetes.

The present study had been carried out in the department of Ophthalmology, M.L.B. Medical College, Jhansi, to search out the extent to which eyes become victim of diabetes mellitus. The cases were selected randomly from Diabetic clinic of department of Medicine and from Ophthalmology department. Details of the findings of each patient was recorded along with general information of patient along with duration of diabetes, duration of treatment, type of treatment whether insulin or non-insulin dependent, all patients went through complete ophthalmic examination and complete relevant investigation viz. Tonometry (Shiotz), slit lamp examination and visual field charting when and where required. Fundoscopy by direct ophthalmoscope by dilating pupils by 10% phenyl ephedrine drops.



In the present study 106 diabetics were examined in which 64 (60.37%) cases were males and 42 (39.62%) were females.

Maximum number of patients were from the age group of 41 - 60 years (52.83%) followed by 21 - 40 years (22.64%) and above 60 years (20.75%).

Type - I IDDM comprised of (26.42%) cases and type - II NIDDM (73.58%).

Incidence of ocular involvement among 106 diabetics was 89.62% and was related to duration of diabetes, rather than with the age of patients. With duration of less than 5 years 20% involvement and 6-11 years (65.22%) and 11-15 years 75% and 16-20 years 90% and above 21 years had nearly 100% involvement in one way or other.

Involvement of ocular adnexia was in 26 cases. Blepharitis was observed in 8 cases, Recurrent sty in 11 cases, Trichiasis in 4 cases, and one case each in ptosis, Acute and Chronic dacryocystitis.

Non specific findings related to conjunctiva were conjunctivitis in 3 cases, xerosis in 2 cases and pterygium in 1 case.

Cornea got involved in 30 cases i.e. decreased corneal sensitivity in 15 cases, which is significant

finding among other changes seen in cornea. Superficial punctate keratitis and corneal opacity in 5 cases, corneal striate in 4 cases and one case of corneal ulcer.

Iris was involved in 19 cases, acute iridocyclitis in 4 cases, pigmentary changes in 3 cases, iris atrophy in 2 cases, rubeosis iridis in one case. Pupillary reaction to light was found sluggish or fixed in 9 cases.

Lenticular changes were more common in females out of total 74 cases there were 23 females and 14 males. Another interesting finding was that the lenticular changes was seen in all the age groups, and it appeared earlier in diabetics and was important factor for visual impairment.

Intraocular pressure was recorded in 86 cases in which fundus was visible. It was then compared in different grades of retinopathy. The mean intraocular tension in eyes without retinopathy  $17.93 \pm 1.0$  mmHg. Mean intraocular tension in eyes with retinopathy was  $18.95 \pm 2.38$  mmHg, this when compared to the eyes without retinopathy was towards higher side. Mean intraocular tension in grade-I retinopathy was  $19.12 \pm 1.82$ ; in grade II  $19.95 \pm 1.91$ ; and finally in grade III, the mean intraocular tension  $15.49 \pm 1.50$  showed lower value than other grades of retinopathy including eyes without retinopathy.

Apart from all these changes we also studied the relation between the development of retinopathy and duration of disease leaving aside the age of the patient, and the severity of disease. The retinopathy seems to develop of any grade on an average of 16.7 years of duration.

With duration of diabetes less than 5 years, there was no retinopathy, with duration of diabetes upto 10 years 16.66% cases had no retinopathy 75% had grade-I retinopathy; and 8.33% cases had grade II retinopathy and none had grade III retinopathy.

With duration of diabetes, upto 15 years 16.6% cases had grade-I retinopathy, 83.33% grade II and none had grade III retinopathy.

With duration of diabetes upto 20 years none had grade - I retinopathy, 70% cases had grade-II and 30% cases had grade III retinopathy.

With duration of diabetes upto 25 years none had grade-I retinopathy, 60% cases had grade II and 40% cases had grade III retinopathy.

With duration of diabetes more than 25 years all cases had only grade III retinopathy.

These findings clearly suggests that as the duration of diabetes increases, the severity of diabetic retinopathy also increases.